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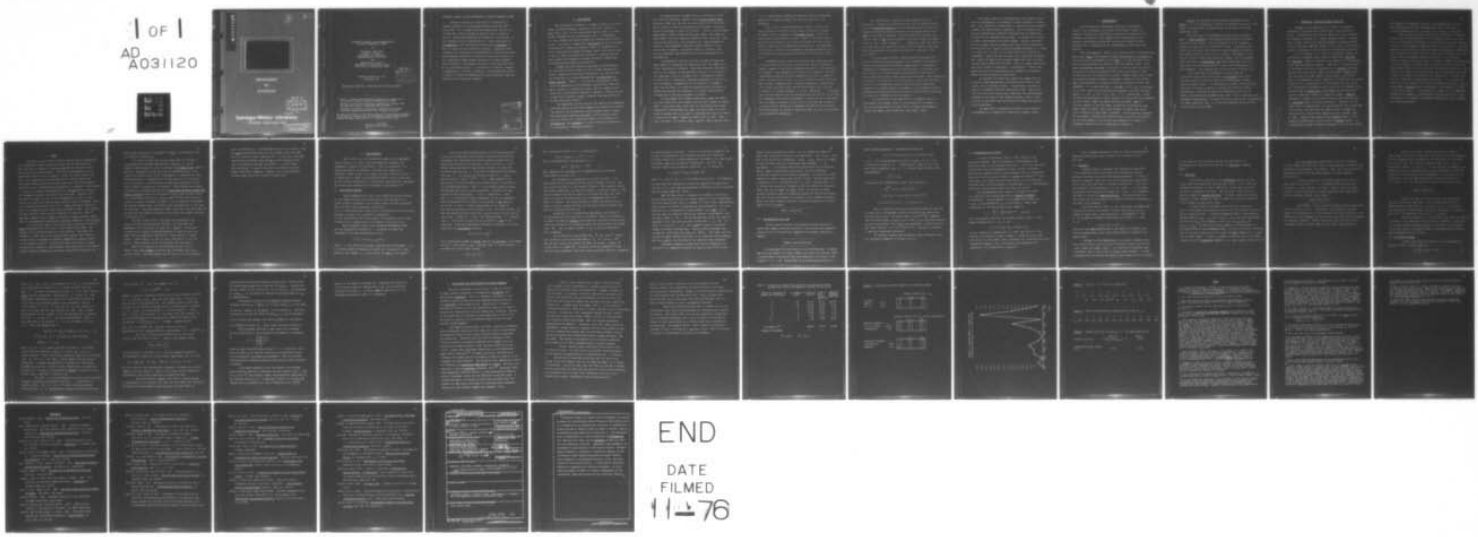

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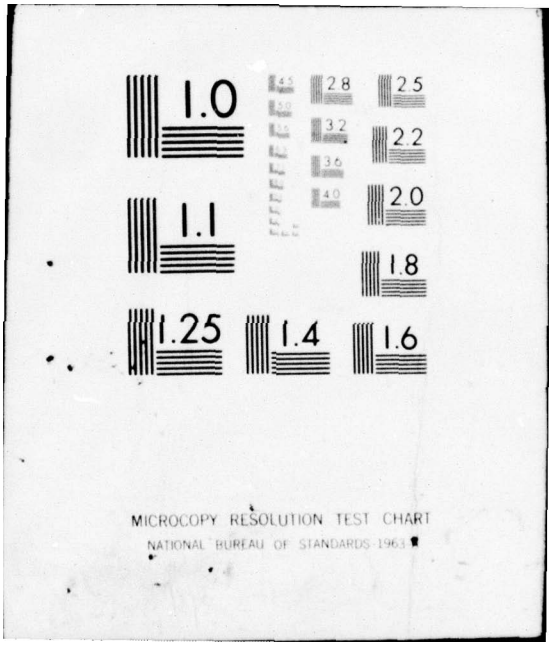
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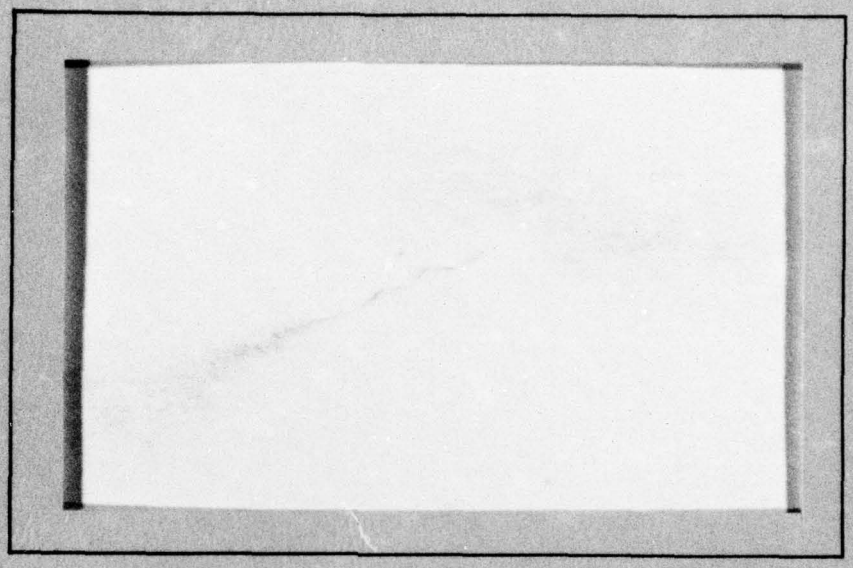

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STOCHASTIC MODELS OF THE DISTRIBUTION OF DYADIC WARFARE IN TIME*

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STOCHASTIC MODELS OF THE DISTRIBUTION OF DYADIC WARFARE IN TIME

Stochastic models are constructed to illuminate the dynamic incidence of international warfare during the 1816-1965 period. It is argued that the probabilistic structure is revealed most clearly through an analysis based on dyads of nations, thereby disassembling such large wars as World War II. The conceptual focus is maintained on a clear delineation of heterogeneity over time and over actors and of contagion in both its addictive and infectious varieties. Departures from randomness are considered as modifications of the Poisson process. Methodological attention is directed at statistical analysis of the interarrival times between initiations of dyadic warfare. Cyclic behavior is investigated tnrough a cosine wave-form variant of the Poisson process. A conclusion of infectious behavior is supported by a variety of analyses. An autoregressive model of order 4 is found to adequately fit the interarrival times and account for the infectious behavior.

1. THE PROBLEM

The initiation of warfare is a dramatic event on the world scene, absorbing attention, resources, and lives, often on a grand scale. War , in the conventional Western view, is a cataclysmic breakdown of the international order, a catastrophe; i.e. war between two nations becomes the ultimate dyadic interaction. Its onset is fraught with uncertainty as the basic unpredictability of timing, participants, number of casualties and level of destruction becomes strikingly clear. The study of war, in all its parts, has proved fascinating for centuries and has been pursued with diligence from many different perspectives, including the most formal and theoretical.

Perhaps the most effective beginning on understanding the conditions of war through the use of formal mathematical models, tempered by statistical verification, was made by Lewis Fry Richardson in his path-breaking work, The Statistics of Deadly Quarrels (1960)[1]. It has become increasingly evident since the appearance of his work that a particularly effective way of coping, in a theoretical sense, with the complex interactions and uncertainties attendant to warfare is through the use of explicitly stochastic models, formally treating uncertainty through probability theory.[2]

We demonstrate in this article that some of the fundamental structural questions that have commanded the attention of theoretical analysts can be addressed using stochastic models coupled with statistical inference procedures. In particular we shall focus our efforts on a clear delineation of the concepts of heterogeneity and contagion, as useful theoretical constructs in a multi-actor, interactive system.

Heterogeneity and contagion will be considered as deviations from the randomness implied by the Poisson process model. Richardson (1960, pp. 129-142) reported a good fit of the distribution of wars in time to the Poisson process model, using data derived from Wright's (1942) list of wars. Others, including Moyal (1949), Denton (1966), Denton and Phillips (1967), and Singer and Small (1972), have also reported research in this direction. Their research has focused on the alternative of a time-heterogeneous departure from randomness required by cyclic behavior in the periodicity of warfare. Richardson (1960, p. 173 and p. 263) has also evaluated "war-proneness" as one conceptualization of heterogeneity.

A conceptually different tack was followed by Singer and Small (1974) in evaluating the proposition that "war begets war", a basic assertion of contagion. While they term this proposition "part of the folklore of international politics", it is nonetheless important, since if contagion were a determinative factor, it would offer a conceptually important explanation of the occurrence of warfare. Recognition of the influence of contagion would then be useful for prediction purposes about the growth of warfare and motivate the examination of various causal factors stimulating or inhibiting contagion in warfare.

To investigate the contagion proposition Singer and Small use their data set on wars between 1816 and 1965 (Singer and Small, 1972). Using these data allows them "to ask whether those years during which international war began were more likely to be followed by another war in the same or subsequent year than were those in which no war began" (Singer and Small, 1974, p. 279). Their "war vs no-war" 2 x 2 turnover table, however, shows a pattern which

is not a statistically significant departure from an independence hypothesis.[3] Singer and Small thus conclude that war is not contagious.

This is not a surprising finding. Indeed, it could have been predicted on the basis of the analysis presented by Singer and Small (1972) in Chapter 9 of The Wages of War, "Cycles and Periodicity in the Incidence of War." There it was shown that the empirical distribution of the intervals between the onset of wars offered a good fit to an exponential distribution. The exponential distribution is derived from assumptions implying randomness and is consistent with a Poisson process model for the occurrence times of wars.[4]

Although one basic conclusion of Richardson (1960) was that the onset of wars fit a Poisson distribution, some of Richardson's analysis appears to contradict randomness and point in the direction of contagion. This emerged out of Richardson's attempt to construct a mathematical model which would adequately describe the number of nations on each side in a war. In constructing this model Richardson went through a lengthy process of making assumptions, forming these into a model, and then either discarding the model (for a variety of reasons) or testing it against his data. Richardson considered some thirteen theories with the twelfth giving the best fit to the data. The assumptions which made up Richardson's theory XII were essentially that the number of parties on each side in a war was the outcome of a process heavily influenced by geography and modified by infection. This would appear to contradict Singer and Small as well as Richardson's own conclusion of randomness.

The contradiction, however, is more apparent than real. Richardson's finding of infection was based upon an analysis which used the individual nations as units, whereas the data used by both Singer and Small and Richardson which fit the random pattern used wars as the units of analysis. The conclusion which follows from this is that one war does not generate other wars (Singer and Small, 1974, p. 279 , 1972, chapter 9 ; Richardson, 1960, pp. 128-31), but that once a war starts the chances of other nations being drawn into that war may be influenced by infection (Richardson, 1960, pp. 275-86).

It is possible, moreover, that Richardson understated the extent to which wars were enlarged by the contagion of the conflict. Because his models were exceedingly complex and, by contemporary standards, his computational aids rather primitive, Richardson did not include in his final theories (including XII), those wars in which there was a total of more than four participants. This effectively excluded 17 of the 91 wars in his data base, or about 18.7 per cent. Since the 17 excluded wars are precisely those which would have exhibited the greatest amount of contagion, it is perhaps now understandable why Richardson found the process by which parties are added to a war only "modified by infectiousness."

The possibility of wars growing by a contagious process is potentially interesting, since it could account for the peaks of war concentration which have been noted by several investigators (Singer and Small, 1972; Richardson, 1960; Moyal, 1949).

This paper reports an investigation of the extent to which wars are heterogeneous or contagious in their stochastic development. The distinctions between what is reported here and previous research are several and are important: First, the models used here are substantially different from those used by Richardson, in which no time dependency was present. Our models are, as will be shown, time-dependent and hence dynamic. Second, the models we will present below are simpler than those used by Richardson. Not only does this simplicity provide greater parsimony as a virtue in itself, but it allows us to include data not considered by Richardson. Third, the data we shall use in exploring the models will consist of dyads which actually fought each other in international wars between 1816 and 1965. This is a longer time period than that covered in Richardson's investigation.[5] Fourth, this paper will focus on heterogeneity and contagion as conceptual models for the initiation of dyadic war.

In addition this paper will explore and demonstrate the utility of some methodological techniques which have not been widely used in political science. In particular, use will be made of a stochastic model of interarrival times. This is not the first time that inter-arrival times have been statistically analyzed in the study of war (Singer and Small, 1972, p. 205), but this is the first time the interarrival times have been explicitly modelled.

We begin with a conceptual discussion of the nature of heterogeneity and contagion in a multi-actor dynamic system.

2. HETEROGENEITY

The processes of international relations, specifically during crisis situations, are seldom "well-behaved"--events are complex, influenced by a multitude of factors, and subject to considerable variability. This suggests that international crisis behavior, particularly during the confusion and turmoil of international war, is characterized by heterogeneity, rather than homogeneity.

This heterogeneity, however, can evidence itself in two quite distinct ways. First, the stochastic process governing the development of the crisis may be heterogeneous over time, i.e., the evolution of the process may be governed by different rules at different times. This time heterogeneity may appear continuous in form, as when the rate of initiation of conflict accelerates over time, or abruptly in form, as when the rate of initiation of conflict suddenly jumps, due to, say, an unexpected change in weapons technology. Effects of this nature may be accounted for in stochastic models by allowing a process rate parameter, λ, to vary with time, t, perhaps $\lambda = a + bt$ or by building in discrete shifts in the process rate parameter, such that the rate equals λ_1 and λ_2 for $t \leq t_0$ and $t > t_0$, respectively. There is also the possibility of cyclic behavior of the process rate parameter. In fact, Denton (1966) and Denton and Phillips (1967) suggest a cyclical pattern of war with peaks successively increasing in size, while Singer and Small (1972) find evidence to support the conclusion of periodic 20 year peaks in warfare. A basic question addressed in this paper is whether such observed behavior should be attributed to cyclical changes in the process rate parameter or, rather, to contagious behavior as it is delineated in Section 3.

Second, the stochastic process may be heterogeneous over actors, i.e., the probabilistic behavior of different actors in a conflict environment may be different and not governed by the same probability laws.

The first type of heterogeneity requires that an adequate model be time-dependent in the parameters governing the stochastic evolution of the system. Thus the parameters of the stochastic process are made explicitly functions of time. As Ginsberg (1971, 1972a) has indicated, some sources of time heterogeneity, which do not interact with the states or the stochastic development of the process, such as broad socio-historical changes, can be eliminated using the device of operational time which essentially alters the time scale in a nonlinear fashion to obtain time-independence of the parameters. This approach will be illustrated in Section 5.2.[6]

The second type of heterogeneity follows from the realization that we are dealing with a multivariate time series, tracing a cohort of actors as they move stochastically through various possible states. In international relations the actors will typically be nations or groups of nations and hence will differ substantially according to almost any conceptually valid set of factors influencing the phenomena under study. Thus it may well be an inadequate approximation to assume that each of the actors is governed by the same probability laws, i.e., that the parameter values for the various actors are the same.

3. CONTAGION: ADDICTION VERSUS INFECTION

Contagion refers generally to a variety of stochastic dependencies concerning one or more actors in an interactive system. Unfortunately, the term has been used in the literature of stochastic processes in a highly ambiguous manner, so ambiguous, in fact, that it has precluded adequate conceptualization of one of the most important aspects of the study of multi-actors systems (including, of course, those in international relations).

For our purposes it is useful to distinguish between two varieties of contagion, which shall be termed here, addiction and infection. Both are relevant to the analysis of international crises and the growth of warfare. Addiction is a characteristic of individual actors and is exhibited in its positive form when the fact that an actor has taken an action makes it more likely that the actor will take similar actions again in the future. Thus, in international crises, addiction is evident when an initial hostile act by a state means it is more likely that this state will accelerate the frequency and/or hostility of subsequent acts. Addiction may be conditional as when aggressors who meet with success become more likely to be aggressors again. Negative addiction is clearly also possible since an experience may have an inhibitory effect on future occurrences. Infection, on the other hand, is characteristic of a group process, and is exhibited when the fact that one actor has taken an action changes the probability of a second actor taking an action (Coleman, 1964, p. 299). Infection may therefore have positive impact on the behavior probabilities of other actors or negative impact. In international conflict infectious contagion is not at all unlikely.

For example, the engaging in hostile interchange within one dyad may significantly increase the probability that a third party initiates warfare with one (or both) of the nations in the dyad.[7]

The distinction between addiction and infection is important on more than conceptual grounds for, as Taibleson (1974) has noted, there is considerable difficulty in statistically sorting out whether a process is heterogeneous among the actors or is addictive. Taibleson suggests that to verify an addictive effect by looking at a sample and then looking to its future to see if prior events correlate with later events, does not determine whether the effect is true addiction (where the fact of an earlier event's occurrence changes the probability of the occurrence of a later event) or spurious addiction (where the fact that an event did or did not occur at an earlier time to an actor changes the estimate of the probability that the individual came from a lower or a higher risk stratum of the population). In either case you will always get the appearance of addiction. This statistical difficulty is not operative in a model assuming the infectious variety of contagion, <u>provided</u> data are available on the behavior over time of the various actors. Since infection is likely to be an important mechanism of contagion in international relations processes, it may be possible to distinguish heterogeneity among actors from contagion on a statistical basis.

4. DATA

The data set used in this research results from an examination of international warfare during the period 1816 to 1965 inclusive. The basic unit of analysis is the dyad, composed of two nations which have entered into armed conflict. The use of dyads appears to be an appropriate device for our interests. There are several reasons for this: Most importantly, recording the day on which a particular dyad began fighting provides the basic time record for evaluating the stochastic models incorporating heterogeneity or contagion. Specifically, it allows us to explore heterogeneity over time and actors as well as the contagious concepts of addiction and infection. Further, since the number of dyads in a war is one surrogate for its size, it will reflect the growth over time of those conflicts which do enlarge. From this perspective World War II was not a single event which started in 1939, but a gradual process which grew from 1939 to 1945. Also looking at dyads provides a more realistic approach to large wars, which in important respects were a complex collection of deadly quarrels rather than the confrontation of monolithic coalitions depicted in some accounts.

Our focus upon dyads is also based upon a definition of war as joint belligerent activity, that is, both nations must fight. The wisdom of this definition may not be self-evident: There may be a tendency to think that if one nation attacks another that defense or counter-attack is automatic, therefore suggesting an analysis based on the individual nation. We would only agree that there is a high probability of defense or counterattack, while pointing out that history contains numerous examples of one nation launching an attack and the

other nation involved not resisting.[8] No war, as we typically view it, existed in these cases.

To create our data base we have drawn upon the listing of international wars between 1816 and 1965 compiled by J. David Singer and Melvin Small in their encyclopedic The Wages of War (1972).[9] The basic advantage of using this list, as mentioned above, is the careful and rigorous work of the investigators in compiling the data. Notwithstanding the availability of the data and the care exercised in its production, it was necessary to make some adjustments in the data to make it suitable for our purposes. These changes were largely occasioned by a central interest in the dyads which actually fought each other in the war; we are not interested in those dyads in which there is no declaration of war or no actual combat.[10] This decision necessitated a scrutiny of Singer and Small's list of opponents to determine who actually fought whom. Alterations were necessary only in the cases of three wars: The Seven Weeks War (1866), World War I and World War II. In each case the number of dyads was reduced with the largest reduction in World War II.[11]

The day on which each of the dyadic war initiations took place in the 150 year period was recorded. If these data are aggregated over the period of a year (as in Table 1), there were 95 years with no dyads initiating fighting, 27 years in which one dyad initiated fighting, 8 years with 2, 6 years with 3, 3 years with 4, 2 years with 5, 10, and 14, and 1 year with 6, 7, 11, 13, and 40. This produced a total of 209 dyadic war initiations.

Much of the analysis in the following sections will be based on the times between successive war initiations (the inter-arrival times). The time between the i^{th} and $(i+1)^{st}$ war initiation

will be denoted by T_i. A problem which exists in the data is that the exact interarrival times are not available since the times are not recorded in hours, minutes, etc. In any case, recording the data more accurately than the day of war initiation would lead to essentially the same conclusions. In recent dyadic war initiations, it would be possible to ascertain the exact moment hostilities commenced. However, this is the exception as exact times could not be found for early wars.

5. DATA ANALYSIS

This section uses the interarrival times and the aggregated data of Section 4 to discuss the heterogeneity, contagion, and randomness of dyadic war initiations. Since the hypothesis of a random distribution of warfare in time has been supported by Richardson (1960) as well as by Singer and Small (1972 and 1974), it is appropriate to begin by assessing the extent to which these data fit the Poisson distribution, a distribution consistent with randomness and inconsistent therefore with contagion or heterogeneity.[12]

5.1 The Poisson Process

Three assumptions are basic to and imply the Poisson process:
(1) The number of dyadic war initiations in one time period is a random variable independent of the random number of dyads initiating war in another (non-overlapping) time period.
(2) In a sufficiently short time period, the probability of two or more instances of dyadic war initiation is negligible.
(3) For sufficiently short time periods, the probability that a dyad initiates war is proportional to the length of the time period.

These assumptions lead to the following mathematical model for the probability of $k(k=0,1,2,\ldots)$ instances of dyadic war initiation in the time period from t_0 to t_1:

$$e^{-\lambda(t_1 - t_0)} \, [\lambda(t_1 - t_0)]^k / k!,$$

where e is the base of the natural logarithm and the parameter $\lambda > 0$ is the mean number of instances of dyadic war initiation in a time period of unit length (λ is also called the rate in the sequel).

One of the characterizing features of the Poisson process is that the interarrival times between successive occurrences are independent and identically distributed random variables with an exponential probability distribution. Thus if we let T be the random variable giving the time interval between one dyadic war initiation and the next, T would have an exponential distribution with mean λ^{-1}. The probability density function of T would then be given by $f_T(t) = \lambda e^{-\lambda t}, \quad t > 0$. Some insight to the implications of this fact about the Poisson process is provided by the following derivation: View T as the random variable giving the waiting time until the next occurrence. Subdivide the time interval of any fixed length t into n equal parts. Given that n is sufficiently large, assumptions (2) and (3) guarantee that the probability of at least one occurrence in <u>any</u> specific one of the n parts is nearly proportional to the length of the interval and hence can be written as $\lambda t/n$ for some constant $\lambda > 0$. Then the probability that there will be <u>no</u> occurrence in any specific one of the n parts is $1 - \lambda t/n$. The random variable T is greater than t if and only if there are no occurrences in <u>any</u> of the n intervals. Given the independence affirmed by assumption (1) we can then write the <u>approximate</u> equality,

$$P(T > t) \approx \left(1 - \frac{\lambda t}{n}\right)^n .$$

This relationship becomes an <u>actual</u> equality <u>in the limit</u> as the length of the n parts goes to zero. Letting, then, n go to infinity we obtain by a basic limit theorem,

$$P(T > t) = e^{-\lambda t} .$$

The distribution function of T is then given by

$$F_T(t) = P(T \leq t) = 1 - e^{-\lambda t}, \quad t > 0,$$

and, by differentiation with respect to t,

$$f_T(t) = \frac{d}{dt} F_T(t) = \lambda e^{-\lambda t}, \qquad t > 0.$$

This completes the derivation of the asserted form of the probability density function of T.

One consequence of assumption (3) is that the rate λ at which dyadic wars are being initiated does not change over time. This could pose difficulties in the present research in two respects: yearly and secular variations. In the first respect, Richardson (1960, p. 129) notes a tendency for wars to begin in the spring and fall, but since years are used as the time period, for the aggregated data, this effect is removed. The second problem of secular variations across a number of years is more difficult and does pose certain problems which will be discussed in Section 5.2.

Assumption (2) is violated when simultaneous occurrence of the initiation of two or more wars is possible. Thus it is illadvised to model the number of nations initiating wars using the Poisson process since any initiation is necessarily dyadic and would violate the assumption. The use of dyads, however, is not in obvious violation of this assumption.

Let us denote the time between the i^{th} and $(i+1)^{st}$ dyadic war initiation in the period 1816-1965 as T_i for $i=1,2,\ldots,208$. Then we can consider the ordered (in time) set $(T_1, T_2, \ldots, T_{208})$ as a time series of length 208. As stated above the assumptions of the Poisson process guarantee that these random variables should be independent

and exponentially distributed. A test of the Poisson assumptions can be made by determining if these random variables are independent. A standard method for testing independence of elements of a time series is through sample autocorrelation coefficients. The k^{th} lag (Pearson) sample autocorrelation coefficient r_k is defined by

$$r_k = \sum_i (T_i - \bar{T})(T_{i+k} - \bar{T}) / \sum_i (T_i - \bar{T})^2,$$

where $\bar{T} = \sum_i T_i/n$ and n is the length of the series. If long(short) waiting times are immediately followed by long(short) waiting times, the first (i.e., k=1) lag autocorrelation coefficient should be positive. Similar remarks apply for k=2,3,... .

When the time series is assumed to be normally distributed, Pearson lag autocorrelations are usually employed to test independence. Since the waiting time series is thought to be more nearly exponentially distributed, we instead use Spearman lag autocorrelations. As in the familiar test of independence using Spearman's rho, ranks of the waiting times are employed, making this approach non-parametric (Cox and Lewis, 1966, p. 166). For the waiting time series, the value of Spearman's first lag autocorrelation coefficient is $r_1 = .269$, for which the p-value is $< .001$. This suggests positive correlation between successive waiting times. Similar positive Spearman autocorrelation coefficients are found for k=2,3,4. This shows that the process of dyadic warfare initiation is not consistent with randomness and also suggests the direction of the deviations from randomness.

Although it appears unlikely that Poisson assumptions are satisfied, for comparative purposes we report a test of fit of the

number of war initiations per year. Let X_i denote the number of dyadic war initiations observed in the year $1815 + i$ for $i=1,2,\ldots,150$. Then under the Poisson assumptions, $X_1, X_2, \ldots, X_{150}$ should be a sample of size 150 from a Poisson distribution with parameter λ. By estimating λ an estimated theoretical distribution can be obtained. Table 1 reports the observed distribution, and the theoretical distribution implied by the Poisson assumptions. An inspection of the differences clearly reveals that the fit is poor. The chi-square test of goodness of fit ($\chi^2 = 169.29$, $DF = 4$, $p < .001$) confirms this. It is evident that unlike the instances of war (as defined by Richardson (1960) and also by Singer and Small (1972)) the process of dyadic warfare initiation cannot be adequately modeled by the Poisson process. The next sections investigate the adequacy of more complex stochastic models for dyadic warfare initiation including formulations of heterogeneity and of contagion.

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Table 1 about here

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5.2. Heterogeneity over time

As was noted in Section 2 the observed cyclical patterns of warfare may suggest wave-like variation in the Poisson rate parameter. Certainly this is a possibility suggested by the graph displayed as Figure 1.

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Figure 1 goes about here

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We investigate this possibility in the following way: In agreement with the analysis of Denton (1966), and Denton and Phillips (1967) a Poisson model is postulated with the probability of an event in the interval t to $t + dt$ proportional to an oscillating function of t

with increasing amplitude. Consistent with this let

$$\lambda(t) = (c + dt)(\cos \gamma t + 1) = 2(c + dt)\cos^2(\gamma t/2),$$

$c, d > 0$, the one being added to insure $\lambda(t) \geq 0$. If X_i is as in Section 5.1, then it is known (Parzen, 1962, p. 125) that under these assumptions that X_i is a Poisson random variable with rate parameter

$$\int_i^{i+1} \lambda(u)\,du.$$

Carrying out the integration we find that the rate is

$$2\int_i^{i+1} (c + ud)\cos^2(\gamma u/2)\,du =$$

$$c + di + d/2 + d(\cos \gamma(i+1) - \cos \gamma i)/\gamma^2 +$$

$$((c + d(i+1))\sin \gamma(i+1) - (c + di)\sin \gamma i)/\gamma .$$

Table 1 reports the results of fitting this model of the data on dyadic wars: The agreement between the observed and theoretical distributions is poor ($\chi^2 = 39.06$; DF = 4; $p = 7 \times 10^{-8}$).[13] This suggests that the distribution of dyadic wars in time is not adequately modelled by the Poisson process with cosine time dependency. An alternative to this model would be $\lambda(t) = e^{c+dt}[\cos \lambda t + 1]$, but this is unlikely to make any substantial difference.

We therefore examine some alternative models consistent with the conceptual framework of Sections 2 and 3.

5.3 Heterogeneity over actors

As stated by Taibleson (1974, p. 878) "there has been consensual agreement that the assumption of heterogeneity over actors will lead to the Greenwood-Yule model". This model specifies a particular form of heterogeneity in that a number of Poisson processes are operative and the rate parameters are not necessarily equal and, in fact, vary according to a gamma distribution.[14] Thus, according to this model the individual nations in the system have different rates of participation in warring dyads (i.e., heterogeneity), unlike the Poisson process where the rates are uniform.

The Greenwood-Yule model leads to the number of events in a specific time interval given by the negative binomial distribution. If the parameters of the gamma distribution are α and β, the probability p_k of obtaining k dyadic ware initiations in any time period of unit length is

$$p_k = \binom{k+\alpha-1}{k} \beta^k/(\beta+1)^{k+\alpha} \qquad k=0,1,2,\ldots \ .$$

For a derivation of this fact see Johnson and Kotz (1969, p. 25). Moment estimates for β and α are

$$\hat{\beta} = (s^2 - \bar{x})/\bar{x} \quad \text{and} \quad \hat{\alpha} = \bar{x}^2/(s^2 - \bar{x}).$$

In this example the estimates are $\hat{\alpha} = .1255$ and $\hat{\beta} = 11.04$. The expected distribution is shown in Table 1. While the fit of this distribution is much better than the Poisson, the differences are still substantial enough so that statistically the differences are significant ($\chi^2 = 19.98$, DF = 4, $p = 5 \times 10^{-4}$).

Thus, although allowing for different rates of participation among the warring dyads gives a better fit, the model is still deficient.

5.4 Addiction

A general model to represent the situation of addiction[15] is to assume that each of the dyads follow independent Poisson processes each with a possibly time-varying parameter. If there is positive (negative) addiction, the rate of a particular dyad should go up (down) each time that it initiates hostilities. If the rates are assumed to vary according to $\lambda = a + bj$ where j is the number of previous times the dyad has fought and a and b are parameters, the model is called the Eggenberger/Polya. This model allows for the possibility of both positive and negative addiction--depending on the sign of the parameter b.

Arbous and Kerrick (1951, p. 411) showed that the Eggenberger/Polya model leads to the negative binomial distribution for the number of dyadic wars in any time period. The parameters α and β of the negative binomial distribution are related to a and b by

$$\alpha = a/b \quad \text{and} \quad \beta = e^b - 1.$$

The fit of the Eggenberger/Polya is then exactly the same as that of the Greenwood-Yule, which from the discussion in Section 5.3 we know is poor.

Although the Polya/Eggenberger is a natural contagious model for certain fields, such as accident statistics (Arbous and Kerrick, 1951 ; Bates and Neyman, 1952), its restrictive assumptions make it doubtful that it can be used to explain dyadic warfare. It seems reasonable that the rate of the various dyads should also be a function

of the number of wars initiated recently by other dyads. To investigate this conjecture, we now turn to infectious contagion models.

5.5 Infection

In contrast to addiction, in an infectious process the rates of all dyads may change when some dyad goes to war. If the rates are increased (decreased), at least temporarily, we say that the process has positive (negative) infection. Most and Starr (1976) call this positive (negative) spatial diffusion. If there is positive infection, long(short) times between the start of dyadic wars should be followed on the average by long (short) times.

To statistically investigate the possibility of infection we use a contingency table approach based on the time between successive entry of dyads into war. A two-way contingency table is constructed by picking numbers $a_1, a_2, \ldots, a_r$ with $0 = a_0 < a_1 < \ldots < a_r$, and then classifying the point (T_i, T_{i+1}) as belonging in cell (j,k) if $a_{j-1} < T_i \leq a_j$ and $a_{k-1} < T_{i+1} \leq a_k$. An infectious process suggests a certain pattern to the cell entries in the contingency table: If there is positive infection, most of the pairs should fall on (or near) the main diagonal (i.e., upper left to lower right). If there is negative infection, most of the pairs should fall on the transverse diagonal (i.e., upper right to lower left).[16]

The Polya/Eggenberger model implies that the difference in time between successive wars should be approximately independent and should have an exponential distribution. Thus, each of the rows of the contingency table should have approximately the same frequency distribution.

In order to make the cell entries large and to have the same number in each row, a 3×3 contingency table was used with divisions 0, 1-100, and ≥ 100 (in days). Table 2 shows the number observed in the 3 intervals, the number expected under independence, and the difference between these numbers.

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Table 2 about here

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The standard χ^2 test indicates deviations from independence ($\chi^2 = 11.3$; DF = 4; $p = .02$). Furthermore, the differences strongly suggest the deviations are in the direction of positive infection. The differences on the diagonal are all positive, and those off the diagonal are typically negative. Similar conclusions would be reached with contingency tables of other dimensions and other division points.

There is no reason to assume that only the length of time between the two most recent wars influences the Poisson parameters. If these parameters are influenced by the difference in time between the $(j-1)^{st}$ and j^{th} previous outbreak of war, there should be correlation between T_i and T_{i+j}. We use the same contingency table analysis to test for independence and, more importantly, to find the direction of the deviations from independence. A chi-square test of independence using the same methodology as above was carried out for $j=2,3,\ldots,9$. The chi-square and p-values are shown in Table 3.

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Table 3 about here

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These tests show that more than just the time between the last two wars are important. The chi-square statistics are significant at the 5% level for $j=1,2,4,5,$ and 7. Furthermore, the same pattern, characteristic of positive infection, is evident in each case. The dependence dies out as j increases.

From this analysis it is evident that some positive infection is present in the system and that more work is necessary in modelling it in order to more precisely capture the character of the process. In the next section a parametric model is developed which provides a quite acceptable fit to the data.

5.6 An Infectious Model

A model that allows for infection is the autoregressive process of order p (AR(p)) given by

$$U_k = \sum_{i=1}^{p} \phi_i U_{k-i} + \delta + e_k, \qquad (1)$$

where $\phi_1,\ldots,\phi_p,\delta$, and p are parameters and the e_k are independent and identically distributed (i.i.d.) random variables each with mean 0.[17] We model the interarrival times T_k as an AR(p) process after using the logarithmic transformation $U_k = \log(T_k + .5)$. The logarithmic transformation is known to be beneficial in modelling series when the percentage change is more homogeneous than the actual change.[18] If the k^{th} waiting time T_k was recorded as 0 days, the actual time was some fraction of a day. Thus, a better approximation (on the average) to the true waiting time is obtained by using .5 instead of 0. Since the transformation $\log(T+c)$ is only sensitive to the value of c $(0<c<1)$ for small T, we use $c=.5$. Thus, the assumed model is

$$\log (T_k + .5) = \sum_{i=1}^{4} \phi_i \log(T_{k-i} + .5) + \delta + e_k. \quad (2)$$

In the case $p=0$, the model (2) can be written

$$\log(T_k + .5) = \delta + a_k, \quad (3)$$

where a_k are i.i.d. random variables with mean 0. This model implies that the variables $\log(T_k + .5)$, hence T_k, are i.i.d. random variables. Since the Poisson assumptions imply that the waiting times T_k are i.i.d., the AR(0) process agrees with the Poisson assumptions. Although the qualitative statement that the Poisson assumptions do not hold has been clearly demonstrated, introduction of model (3) is useful in determining the amount of infection in the system since it represents a baseline case.

The order p of the autoregressive process can be estimated through the partial autocorrelation function. If the model is actually AR(p), for $j>p$ the j^{th} order (estimated) partial autocorrelation η_j is approximately normally distributed with mean

0 and variance n^{-1}. Thus, for example, for $j > p$

$$P(|\eta_j| > 2.85n^{-\frac{1}{2}}) = .004.$$

Suppose we take the reasonable position that we are willing to consider only p such that $p \leq m$, where an order larger than m would not be practical or lack plausibility. The backward method of selecting the order of the process is accomplished by picking positive constants c_j for $1 \leq j \leq m$ and then picking p to be the largest j such that $|\eta_j| > c_j$. The constants c_j are picked such that if model (3) is true, the probability of obtaining any significant partial autocorrelation $(1 - \prod_1^m P(|\eta_j| \leq c_j))$ is small. Suppose we pick $m=12$ and $c_j = 2.85n^{-\frac{1}{2}}$ for $1 \leq j \leq 12$. Then the level of significance of the backward method is approximately $\alpha = 1 - (.996)^{12} = .05$.

The first 12 partial autocorrelations for the interarrival times are shown in Table 4. Based on the backward method

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Table 4 about here

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discussed above, we estimate $p = 4$. The least squares estimates of the parameters of model (2) (using natural logarithms) with $p = 4$ are

$$\hat{\phi}_1 = .269,\ \hat{\phi}_2 = .132,\ \hat{\phi}_3 = .068,\ \hat{\phi}_4 = .193, \text{ and } \hat{\delta} = .777.$$

The fact that all four autoregressive parameter estimates are positive suggests positive infection. That is, this model implies long(short) interarrival times follow long(short) interarrival times.

The above analysis suggests that there is some advantage in using model (2) to forecast time until the next dyadic war initiation. To quantify the additional accuracy obtained, we assume that the

forecasting loss function is given by square error. Table 6 gives the minimum mean square error predictor of $\log(T_{n+1} + .5)$ given the previous values of the series (i.e., T_i for $1 \leq i \leq n$). The forecasts for $\log(T_{n+1} + .5)$ can easily be converted into predictions for T_{n+1} by exponentiation.

The forecast accuracy can be measured through the model residuals given by $\hat{e}_k = \log(T_k + .5) - \hat{\sigma} - \sum_1^4 \hat{\phi}_i \log(T_{k-i} + .5)$ for model (2) and $\hat{a}_k = \log(T_k + .5) - \sum_1^n \log(T_i + .5)/n$ for model (3). The forecasting error of the next value of $\log(T_{n+1} + .5)$ can be estimated by the residual mean square error given by $\sum_5^n \hat{e}_k^2/(n-4)$ for model 2 and by $\sum_1^n \hat{a}_k^2/n$ for model (3). These values are given in Table 5.

The percentage reduction in mean square error obtained by the introduction of the autoregressive parameters is estimated by

$$r^2 = 1 - \frac{\sum_5^n \hat{e}_k^2/(n-4)}{\sum_1^n \hat{a}_k^2/n}.$$

Using the data from 1816-1965 we find $r^2 = 1 - 7.105/9.165 = .225$. Thus, we obtain a 22.5 percent reduction in forecasting variance by introducing the autoregressive parameters. This 22.5 percent reduction gives a numerical measure of the extent to which infection is present.

A chi-square goodness of fit test based on the residual autocorrelation function is available (Box and Jenkins, 1970, p. 291). If r_k is the k^{th} lag (Pearson) sample autocorrelation coefficient of the residual series $\hat{e}_j$ (i.e., replacing T_i with $\hat{e}_i$ in the defining formula for r_k in Section 5.1), the χ^2 statistic is $Q = N \sum_1^m r_k^2$

where N is the number of residual $\hat{e}_j$. Using m=30 the value of Q is 31.65 which should be compared with a χ^2_{25} null distribution. This gives a p-value of .17, which suggests that the fit of the autoregressive model of order 4 is reasonable.

6. CONCLUSIONS AND IMPLICATIONS FOR FUTURE RESEARCH

The basic conclusion of this article is that the process of dyadic warfare initiation is predominantly contagious, and specifically infectious. This is a statement descriptive of the stochastic process of war and it has important implications for the prediction of the growth of war. Specifically, the model developed in Section 5.6 for the interarrival times can be used to forecast the time period to the next dyadic war initiation. The fit of this model must be considered surprisingly good in view of the discussion in Section 2 of the potential impact of war on international system dynamics.

Such empirical predictions, of course, should be interpreted with considerable caution and work begun on a causal interpretation of warfare in which relevant causal variables would be identified and quantified. Consistent with this and our finding of infectiousness we have a conceptual framework which suggests that one question for further research is why some wars spread and others do not. Rather than asking what causes wars, we are asking what makes some of them grow.[19] We might seek causal variables which influence this growth process by looking to previous research on the contagion of social behavior. Two variables which immediately attract our attention are interaction opportunity and status. The basic concept here is one of centrality (Reynolds, 1971).[20] Midlarsky (1970) found among Latin American nations a strong relationship between a nation's diplomatic status and the diffusion of military coups. A substantial amount of research in other areas indicates that the behavior of high status individuals is associated with subsequent diffusion of that behavior (Rogers and Shoemaker, 1971).

There is also research which supports the proposition that contagion within a population is strongly associated with the extent of interaction within the population. Coleman's research on the diffusion, or contagion, of medical innovations indicates quite clearly that physicians who had the greatest interaction with each other were also those who adopted the innovation the most rapidly and extensively (Coleman, Katz and Mengel 1957). In the domain of international politics there are, of course, numerous sorts of interaction processes. To name but a few, geographic regions (Russett (1967)), foreign trade (Alker and Puchala (1968)), as well as cultural bonds and alliances (Deutsch and Singer, 1964) all furnish interaction opportunities and patterns. Alliances, since they reflect political commitments, perhaps provide a template for the future growth of wars. Previous research has focused upon the relationship between various alliances patterns and international warfare (Singer and Small, 1966 and 1968). This research nevertheless has not directly addressed the probability of a war spreading within an alliance network.

Further research might profitably pursue another line of inquiry as well. The data used in the present study represent a rather special conceptualization of international war. It contains a smaller body of data than would have been the case had we chosen to use the complete data set of Singer and Small, or of Wright or Richardson. The use of these data sets would in one way or another led us astray from our focus upon the extent to which violent conflicts of substantial size (at least 1,000 fatalities) occurred in the nation-state system. Richardson's data included numerous

actors such as ethnic, cultural or political groups existing within a nation, and consequently the wars and actors listed were not exclusively international. Wright's list also had the characteristic of including more than international wars. Other listings exist, but these also tend to focus upon more than international wars, as well as being limited to a more contemporary time-span.

While these data collections were of limited use to us in the present study, they do have potential for extending this research. In addition, there exist other lists of wars, generally dealing with more contemporary phenomena, which could be useful in studying the extent to which local conflicts of various types tend to spread and at what rates. For example, Kende's (1971) research on local wars presents not only a listing but a typology as well. Such a list could be helpful in identifying the processes of contagion.

Table 1 Instances of Dyadic War Initiation and Expected Poisson, Poisson with Cosine, and Negative Binomial Distributions

i number of instances of dyadic war initiation	n_i number of years	Poisson Expected	Poisson with Cosine Expected	Negative Binomial Expected
0	95	37.49	66.43	109.76
1	27	51.99	31.69	12.63
2	8	36.05	19.66	6.52
3	6	16.66	12.94	4.24
4	3	5.78	8.34	3.03
5	2	1.63	5.10	2.30
≥ 6	9	.45	5.90	11.52
chi-square (χ^2)		169.29	39.06	19.98
degrees of freedom		4	4	4

$\bar{x} = 1.387$ $s^2 = 16.70$

Table 2. Relationship Between Successive Interarrival Times

		Observed Numbers for T_{i+1}: 0	1-100	>100
Observed Numbers for T_i	0	51	21	21
	1-100	27	18	11
	>100	15	17	25

		Expected Numbers for T_{i+1} Under Independence: 0	1-100	>100
Expected Numbers for T_i Under Independence	0	42.0	25.3	25.7
	1-100	25.3	15.2	15.5
	>100	25.7	15.5	15.8

		0	1-100	>100
Difference Between Observed and Expected	0	9	-4.3	-4.7
	1-100	1.7	2.8	-4.5
	>100	-10.7	1.5	9.2

Figure 1.--Average Number of Dyadic War Initiations per Year for 10 Year Periods

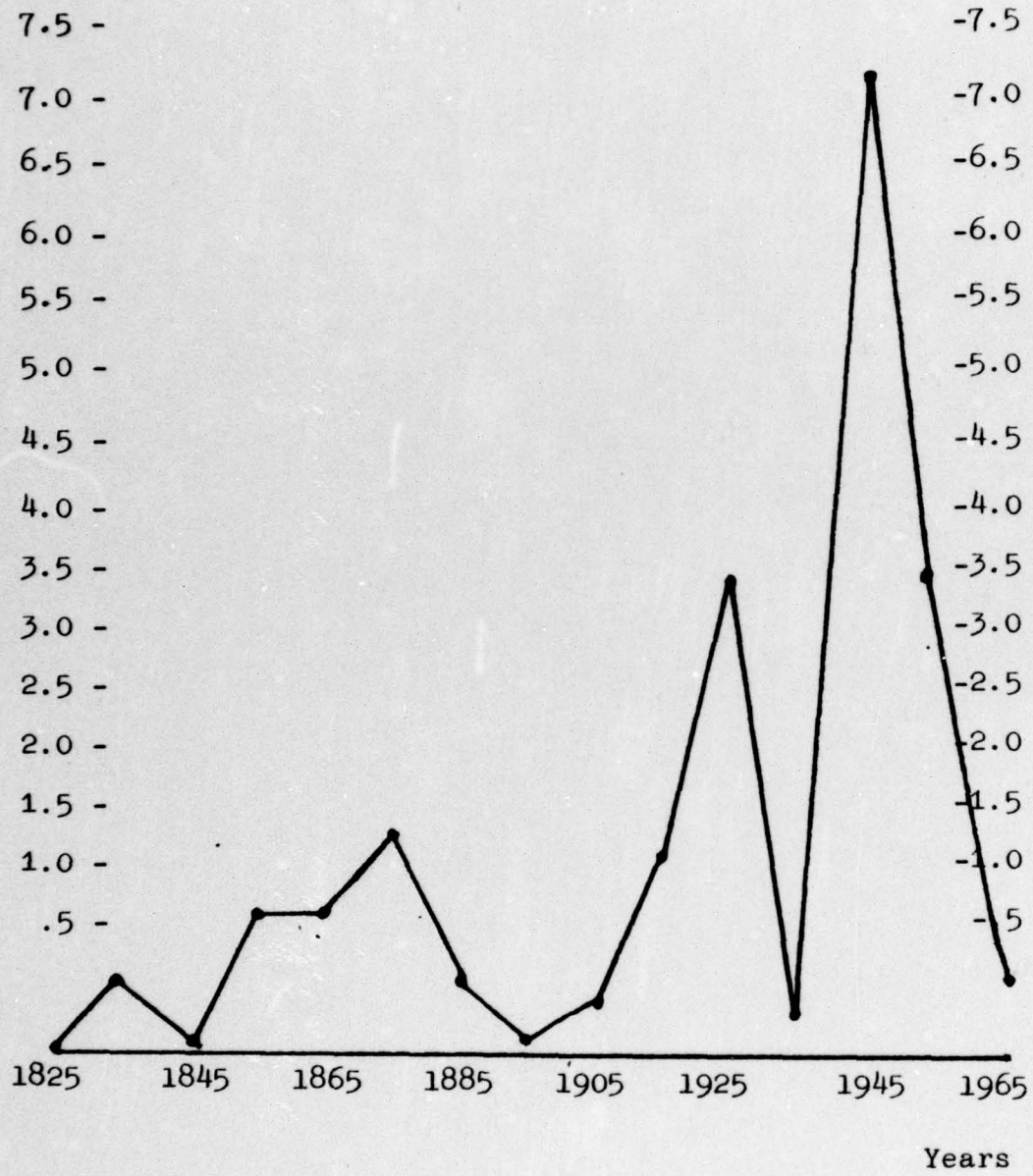

Table 3. Values of χ^2 tests for independence

k	1	2	3	4	5	6	7	8	9
χ^2	11.3	14.1	6.9	31.5	11.6	4.8	10.5	6.4	4.6
p	.02	.007	.14	2×10^{-6}	.02	.31	.03	.18	.33

Table 4. Partial Autocorrelation Coefficients for $\log (T_i + .5)$

k	1	2	3	4	5	6	7	8	9	10	11	12
η_k	.319	.167	.034	.290	.056	.063	.141	-.172	-.024	.106	.056	.024

Table 5. Optimal Predictor of $\log(T_{n+1} + .5)$ and Mean Square Error

	Model 2	Model 3
Optimal Predictor	$\hat{\delta} + \sum_1^4 \hat{\phi}_i \log (T_{n+1-i} + .5)$	2.059
Forecasting (mean square) error	7.105	9.165

NOTES

[1] A review of Richardson's efforts in this area will not be presented here since an extensive description of the twelve models is presented in Zinnes (1975 and forthcoming). A more general evaluation of the Statistics of Deadly Quarrels may be found in Wilkinson (1976).

[2] For a speculative discussion of this see Duncan (1976).

[3] When the data are divided into Nineteenth and Twentieth Century periods, the resulting tables still do not suggest dependence.

[4] The Poisson process and its implications will be discussed in Section 5.1.

[5] Although the Statistics of Deadly Quarrels was published in 1960, much of the research it contains was completed between 1941 and 1949; Richardson died in 1953.

[6] Other sources of time heterogeneity, which arise from the feedbacks between the phenomenon under study and other social, economic, military, or political processes, are not so easily handled. Thus, for example, in a study of a long term conflict environment such as the Mideast, it must be realized that a major event such as a war may have serious and profound consequences for the social, economic, military, and political processes of the system. The fact that warfare changes the factors which determine the future incidence of warfare means not only that the stochastic process in operation is not homogeneous in time, but that actual realizations of the process determine its future development. Such heterogeneities cannot be dealt with simply by letting the parameters be functions of time and resorting to more computationally involved estimation procedures. Usually the only recourse for the analysis of processes this complex is to large-scale simulation methods on a computer. These are relatively easy to implement in specific situations.

[7] Most and Starr (1976), writing in the context of a study of the spread of warfare, have also noted the importance of drawing a distinction between these two types of contagion. They have labelled a process displaying addiction, a reinforcement process, while a process displaying infection is called a spatial diffusion process.

[8] Examples of this are to be found in Germany's attacks on Czechoslovakia in 1939 and Denmark in 1940, as well as the annexation of Austria in 1938. Further, the Soviet invasion of Estonia, Lithuania and Latvia in 1939 found no resistance. More recently India's annexation of Goa was not resisted by Portugal.

[9] For a discussion of Singer and Small's criteria for including an international war in their listing, see pp. 17-39 of their report (1972).

[10] To illustrate the potential of this problem, had we accepted Singer and Small's dyads it would have necessitated the recording of the United States and Finland as a warring dyad in 1942 when Finland attacked the Soviet Union. However, in World War II the United States

and Finland not only failed to declare war on each other, but maintained diplomatic relations until 1944.

[11] Judgments as to who actually fought whom were substantially aided by Richardson's (1960) matrix of each war. Wright's (1965) listing was also used, but inaccuracies were found in several dates. For example, Wright (p. 1537) lists Bulgaria's declaration of war against the United States in World War II as June 22, 1941, almost six months prior to Pearl Harbor! Bulgaria's actual declaration of war against the United States took place on December 13, 1941. Further, Wright recorded many members of the British Empire as entering World War II in January, 1942 when in fact these acts were taken in December, 1941 (Royal Institute for International Affairs, 1947).

[12] For other research in international politics which relies upon the Poisson process, see, e.g., Job (1973), Siverson and Duncan (1976), and McGowan and Rood (1975).

[13] If Y has a chi-square distribution with DF=4, the exact p-value can be obtained from the equation,

$$P(Y>\chi^2) = (1 + \chi^2/2)e^{-\chi^2/2}.$$

[14] The gamma distribution specifies a positive random variable, say X, whose probability density function is proportional to $x^{\alpha-1}e^{-x\beta}$ where α and β are positive parameters.

[15] Most and Starr call this phenomenon reinforcement.

[16] An analogous procedure to one suggested by Most and Starr (1976) could also be used. That is, break the time period up into two non-overlapping periods and for each dyad record the number of times they fought in the two periods. These observations can be labelled (U_i, V_i), $i=1,\ldots,n$, where n is the number of dyads. A two-way contingency table analysis could be used on the pairs (U_i,V_i) as in Most and Starr. A problem with this approach is that many nations do not maintain their national status over the entire time record because of changes in national boundaries, annexations, mergers, etc. This problem becomes more severe as the time span studied increases in length.

[17] For a more complete explanation of the methodology used in this section see Box and Jenkins (1970) or Nelson (1973).

[18] Acton (1959; p. 223) has written "Data that are counts of populations, vital statistics, census data, and the like are almost always improved by taking logs...Charles Winsor frequently prescribed the taking of logs of all naturally occurring counts (plus one, to handle that embarassing quantity zero) before analyzing them--no matter what the sources [of the data]". Tufte (1974; p. 108) has suggested several reasons why one might use a logarithmic transformation in regression studies; an important one for our data is "Badly skewed distributions--in which many of the observations are clustered together combined with a few outlying values on the scale of measurement--are transformed by taking the logarithm of the measurement so that the clustered values

are spread out and the large values are pulled in more toward the middle of the distribution".

[19] This it may be argued is really the implicit question of much research on the causes of war. Many of the variables indicating war are closely associated with the size of war. For example, Singer and Small (1968) operationalize war as a dependent variable by measuring the nation-months, battle casualties and the number of wars begun. The first two are reflective of size.

[20] Singer and Small (1972) have presented data indicating that centrality is not associated with war participation. Their data, however, are of a geographic nature.

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Stochastic models are constructed to illuminate the dynamic incidence of international warfare during the 1816-1965 period. It is argued that the probabilistic structure is revealed most clearly through an analysis based on dyads of nations, thereby disassembling such large wars as World War II. The conceptual focus is maintained on a clear delineation of heterogeneity over time and over actors and of contagion in both its addictive and infectious varieties. Departures from randomness are considered as modifications of the Poisson process. Methodological attention is directed at statistical analysis of the interarrival times between initiations of dyadic warfare. Cyclic behavior is investigated through a cosine wave-form variant of the Poisson process. A conclusion of infectious behavior is supported by a variety of analyses. An autoregressive model of order 4 is found to adequately fit the interarrival times and account for the infectious behavior.